Trauma Rounds

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Inhalation Injury in Burn Patients

F. WILLIAM BLAISDELL, MD:* *The case we have selected for presentation today is a problem of inhalation injury. The case will be presented by Dr. Daniel Capen.*

DANIEL CAPEN:† The patient is an 18-year-old white man who, while under the influence of drugs, fell asleep while smoking and sustained a burn involving both arms. Apparently inhalation injury also resulted because the patient was found to be very short of breath on admission. The medical history was significant: the patient smoked two to three packs of cigarettes a day and had a chronic productive cough.

On physical examination, tachypnea and diffuse wheezes throughout the chest were noted. A 20 percent second and third degree burn of both arms and axillae was apparent.

Initial determination of blood gases included an arterial oxygen pressure (Po_2) of 70 and a carbon dioxide pressure (Pco_2) of 30 mm of mercury. However, the patient became progressively more tachypneic during the course of resuscitation and within an hour of admission the arterial Po_2 fell to 38 mm of mercury. An endotracheal tube was passed and positive pressure ventilation instituted. Following intubation and positive pressure ventilation, the arterial blood gases stabilized on 40 percent oxygen at an acceptable level. Frequent suctioning was required for abundant tracheobronchial secretions, which contained carbonaceous material. No abnormalities were seen on an initial x-ray film of the chest. On subsequent x-ray studies of the chest the progressive development of infiltrates in both lung fields was seen over a period of 24 to 48 hours.

During the next two to three days, values on determinations of blood gases gradually improved and pulmonary infiltrates cleared sufficiently so that the patient was extubated on the fifth day after injury. The respiratory course during the subsequent eight to ten days was complicated by abundant pulmonary secretions which the patient expelled with some difficulty. At the end of this time, respiratory function had returned to normal levels and treatment involved only the routine management of the burns.

STEVEN PARKS, MD:‡ This patient's burns were the least of his problems. He could have recovered sufficiently to be managed out of the hospital relatively promptly had it not been for the inhalation injury.

In the past—20 to 30 years ago—most patients with major burns died because of burn shock.

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TABLE 1.—*Carbon Monoxide Poisoning and Carboxyhemoglobin Levels*

Carboxyhemoglobin Level (Percent)	*Severity*	*Symptoms*
20	Mild	Headache, mild dyspnea, visual changes, confusion
20-40	Moderate	Irritability, diminished judgment, dim vision, nausea, fatigability
40-60	Severe	Hallucinations, confusion, ataxia, collapse, coma
60	Fatal	....

TABLE 2.—*Sources of Noxious Chemicals in Smoke*

Compound	*Noxious Products of Combustion*
Polyethylene, polypropylene	Clean burning, combustion to carbon dioxide and water
Polystyrene	Copious thick black soot and smoke—carbon dioxide and water, some carbon monoxide
Wood, cotton	Aldehydes (acrolein)
Polyvinylchloride	Hydrochloric acid
Nitrogen containing compounds, acrylonitrile, polyurethane	Hydrogen cyanide
Fire retardants frequently produce toxic fumes	Halogens (chlorine, bromine, fluorine), ammonia

Once the mechanism of burn shock was understood and combated with appropriate fluids and monitoring this no longer remained a major clinical problem. The next major cause of mortality was sepsis in the burn wound. However, in the last ten years the development of local therapy for burn wounds (such as silver nitrate, sulfamylon or silver sulfadiazine) has produced a pronounced drop in deaths from wound sepsis. As a result we have been left with respiratory failure as the commonest cause of death. This problem can develop at any time in the course of management. The pulmonary complication rate in most burn units is between 20 and 25 percent with 70 to 85 percent of patients ultimately dying of respiratory failure.

There are many different causes of respiratory insufficiency in burn patients. These include inhalation injury and bacterial pneumonia as the commonest, the latter being the principal late cause of death. Other types of respiratory failure which can occur less commonly include acute pulmonary edema from fluid overload, aspiration pneumonia, pulmonary embolism and the respiratory distress syndrome—which follows all those types of trauma in which there has been shock and extensive soft tissue injury.

Inhalation injury, which I will concentrate on today can produce three different types of problems, all of which are manifest in the immediate postburn period. These are inhalation of heat, inhalation of carbon monoxide and inhalation of noxious chemicals. The heat injury produces damage to the upper air passages only and rarely affects the airway below the level of the larynx. The critical problem is laryngeal edema which can lead to rapid deterioration and prompt death, if not treated vigorously. The initial manifestation of laryngeal injury is stridor. When the airway obstruction becomes critical, immediate access to the airway is indicated by intubation or, if this is difficult, by immediate tracheostomy.

Carbon monoxide poisoning is the second thing you must look for in any case of smoke inhalation. The normal carboxyhemoglobin level of blood is less that 1 percent unless the patient is a smoker in which case it may be 5 to 10 percent. Any carboxyhemoglobin level greater than 10 percent is definitely abnormal, 20 percent is serious and 60 percent is usually fatal (See Table 1). In most cases of smoke inhalation, carboxyhemoglobin levels are between 15 and 25 percent. It is relatively rare for us to see levels higher than this because higher concentrations are usually lethal at the time of the burn. This is the major immediate cause of death in patients injured in fires. Short of lethality, levels greater then 30 percent alter consciousness and lead to confusion or coma. For this reason, when a burn patient is first seen it is appropriate to start administration of 100 percent oxygen immediately and check carboxyhemoglobin levels as soon as possible. The half-life of carboxyhemoglobin is about four hours breathing room air. It is about 30 minutes when the patient breathes 100 percent oxygen. This emphasizes the importance of administering oxygen in these patients without waiting for verification of carbon monoxide poisoning.

Smoke inhalation usually is associated with inhalation of noxious chemicals. These chemicals are produced by the combustion of cotton, wood and especially plastics such as polyurethane and polyvinyl chloride (See Table 2). These toxic substances are oxides of sulfur and nitrogen, hydrochloric and hydrocyanic acids, and various alde-

hydes. Acrolein is an aldehyde produced in high quantities in wood smoke. Ten parts per million of acrolein will produce blatant pulmonary edema. This pulmonary edema results from a combination of chemical damage to the tracheal mucosa, and outpouring of fluid from alveolar irritation. When these chemicals are inspired, ciliary action and mucous transport are immediately depressed. In addition, there is peribronchiolar edema which produces obstruction of small airways. Clinically, this is indicated by wheezing. There may be sloughing of the tracheobronchial mucosa and thick chunks of mucosa may be coughed up which represent actual casts of the lower airway. The patient presented today actually coughed up bronchial casts which included recognizable bifurcations of bronchi. The end result of this complication is usually bronchopneumonia, which is the ultimate cause of death in these patients. The mortality for inhalation injury of the tracheobronchial tree may be as high as 85 percent.

In the patient discussed today there was a chemical tracheobronchitis with peribronchial edema which produced obstruction of the small airways. This caused hypoxia until positive pressure ventilation and administration of increased concentrations of oxygen were undertaken. It was remarkable that the patient recovered rapidly enough for use of a ventilator to be discontinued in five days but it was ten days before recovery to the preburn state. Thermal injury of the upper airway was also present, as shown by the development of a granuloma on one of the vocal cords.

I would like to emphasize that in any case in which there is evidence of singed hairs on the patient's face, eyebrows or hairs of the nose, or burns about the face, there is possibility of inhalation injury even though initial findings on respiratory examination are normal. Patients must be examined at frequent intervals. Shortly after the onset of rales, wheezing or stridor, the patient's course may be rapidly downhill and he may become hypoxic and require emergency intubation. Frequent and repeated physical examinations will give the earliest reliable signs of impending trouble.

CHARLES BLAKELY, MD:* We did not obtain a carboxyhemoglobin level in this patient at the time of admission, but two hours later, after 100 percent oxygen had been administered, there was a carboxyhemoglobin level of 16 percent. In addition, I carried out a bronchoscopic examination immediately after admission. The airway was found to be slightly erythematous, but the magnitude of the injury was underestimated. The extent of damage to the upper airway was not fully evident until several days later. One of the reasons for this was that bronchoscopic examination was done immediately after the patient's arrival and it is probable that sufficient time had not elapsed for the full extent of the injury to be recognized.

DR. PARKS: I believe that in all patients who have evidence of inhalation injury, even mild, fiberoptic bronchoscopy should be done as part of initial assessment at the time of admission. In several series this has been done routinely and it has been shown that possible difficulties can be anticipated rather than waiting for respiratory obstruction or a fall in arterial blood oxygen tension to develop. Erythema and early edema of the larynx and trachea are evidence that inhalation injury has been sustained. Intubation and positive pressure ventilation must be carried out immediately if survival rates are to be improved and complications reduced in such cases.

We use nasotracheal and orotracheal tubes for the initial resuscitation. This is definitive in most patients. In some of the major inhalation injuries, there may be enough mucosal damage so that it is difficult to keep a patient's tracheobronchial tree clear. Although we have preferred to avoid tracheostomy as part of initial management, under these circumstances tracheostomy may be indicated. Tracheostomy is also indicated in other circumstances in which there has been severe upper airway damage with acute laryngeal edema. Those inexperienced with intubation probably should do an immediate tracheostomy rather than attempt to place a tube when respiratory distress develops.

DR. BLAISDELL: *How do you carry out emergency tracheostomy, Dr. Parks?*

DR. PARKS: If you have good light and good exposure and the patient is not in distress, you can do a conventional tracheostomy at the level of the second or third tracheal ring using a transverse incision. If the patient is short of breath or the problem is very urgent, you can do a coniotomy, even with a penknife if necessary. The cricothyroid membrane is easily located by palpation of the extended neck. The thyroid cartilage can be felt and the defect between it and the cricoid cartilage can be easily opened with one transverse

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motion of the knife. It is important, however, to control the knife so that the posterior wall of the trachea is not injured with the initial thrust of the knife. Bleeding is usually negligible as this is an avascular area. An endotracheal tube can be inserted rapidly and control of the airway obtained. This technique can be used for any patient in whom an extremely urgent airway problem is present, including someone who has aspirated foreign material as a result of loss of consciousness. Later, preferably within 24 hours, this should be converted to a conventional tracheostomy.

At San Francisco General Hospital we rarely use emergency tracheostomy because anesthesia expertise is readily available, 24 hours a day. In other hospitals, however, tracheostomy may frequently be indicated. The only problem is that this results in earlier contamination of the airway because the raw tracheostomy wound often heals poorly in critically injured patients and allows contamination of the lower airway. Orotracheal or nasotracheal intubation seems to be associated with a cleaner airway for a longer time.

Our anesthesiologists use sedation, a small dose of curare followed by succinylcholine, and can intubate the agitated patient readily in this fashion. However, one has to be expert in the use of these drugs and I would not advocate this technique for use by the inexperienced.

DR. BLAISDELL: It should be remembered that in a patient who comes in off the street, and who has not been given oxygen and has been breathing ambient air, the administration of 100 percent oxygen by mask usually improves the situation immediately. This permits plenty of time to obtain control of the airway in a conventional fashion.

A PHYSICIAN: *What is the role of steroids in the management of acute airway injury?*

DR. PARKS: Pediatricians have considerable experience with the use of steroids in acute inflammatory airway edema. Edema in burns, however, will not go away with steroid therapy. Such edema is a burn injury as opposed to an infectious injury. While the wheezing from peribronchiolar edema is similar to asthma, it is a chemical injury which is not reversed by steroids. We have tried aminophylline and the other agents used in asthma to treat this type of wheezing but it is not very successful. Bronchodilators do not resolve peribronchiolar edema or mucous plugs but may reduce associated bronchospasm.

Beyond this, steroids are contraindicated in burns because they decrease resistance to infection. Dressler, in a number of experimental studies in animals, found that mortality was increased dramatically in the group of animals that had experimental burns treated with steroids. Although in the animals temporary improvement was achieved using steroids, mortality was doubled in 72 hours. Therefore, I believe that steroids are contraindicated in inhalation injury.

A PHYSICIAN: *Does positive pressure ventilation have any direct role to play besides creating a closed system and increasing the concentration of oxygen?*

DR. PARKS: In several series, one of the remarkable things noted was that positive airway pressure controls the edema. The opposite of this is the observation that creation of a tracheostomy without utilizing positive pressure removes the ability to generate a positive pressure and cough. This has resulted in rapid deterioration and outpouring of pulmonary edema in a number of patients.

Positive pressure ventilation maintains patency of terminal airways and tends to keep them open, preventing progressive atelectasis. When there is any evidence of airway injury in a patient we institute positive airway pressure immediately and believe that by so doing we maintain patency of the airway.

DR. BLAISDELL: *Dr. Parks, would you comment on the effect of burn eschar on ventilation?*

DR. PARKS: Within a matter of hours after a major burn of the chest, the eschar, even though it is not circumferential, will restrict thoracic motion. The patient can become more and more hypoxic and has to work harder to breathe. When the eschar occupies a quarter or more circumference of the chest, immediate escharotomy should be carried out by making multiple longitudinal incisions in the eschar down to viable subcutaneous tissue. It has been our observation that after this is done, patients start to breathe better immediately.

JAMES HOLCROFT, MD:* Dr. Hiram Polk's studies are of interest. He produced inhalation injury in rats and attempted to cause pulmonary edema by administering large quantities of fluids. Surprisingly, he was unsuccessful in producing pulmonary edema despite a dramatic fluid overload. Therefore, the major problem in edema associated with

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inhalation injury would seem to be the direct effects on the airway of the smoke itself.

I am not sure that use of positive pressure ventilation will actually decrease the burn edema. Dr. Robert Demling has studied the effect of positive pressure ventilation on pulmonary edema. While positive pressure ventilation does maintain airway patency, and the values on determinations of blood gases improve, it does not decrease the amount of water or edema fluid in the lungs. This improvement may be the result of ventilation of more alveoli.

GEORGE SHELDON, MD:† I would like to point out that arterial Po_2 determination does not give any indication of the level of carboxyhemoglobin. So, for all practical purposes, it is best to anticipate that carboxyhemoglobin levels must be high and to maintain administration of 100 percent oxygen for one hour or longer whenever there is evidence of inhalation injury. By this time the patient's hemoglobin level should have recovered sufficiently to prevent death. This is particularly important in hospitals where carboxyhemoglobin levels cannot be obtained.

DR. PARKS: The major problem with the co-oximeter used for carboxyhemoglobin measurements is keeping it clean and ready to use. If the machine is meticulously maintained, it will give good results. It is very simple to use and anybody can learn to run it. The primary reason for measuring carboxyhemoglobin is to know when you can safely stop giving high concentrations of oxygen. We should continue to give oxygen until the carboxyhemoglobin level is below 15 percent. By so doing it is possible to avoid prolonged administration of 100 percent oxygen which in itself can produce pulmonary damage.

DR. BLAISDELL: In summary, this discussion emphasizes that if a burn patient dies soon after injury it will most likely be due to a respiratory problem. Other aspects of burn injuries such as fluid resuscitation and prevention of prolonged shock are better understood and managed.

Things can happen very quickly to the airway, as Dr. Parks emphasized, which may result in death. These include direct damage from heat and inhalation of toxic substances, including carbon monoxide. Edema of the airway can develop very rapidly and a patient can change from appearing to be relatively well to being critically anoxic in a matter of minutes. We must assess and continue to reassess the airway in a patient in the initial period following a burn sustained in a closed area. At present, as Dr. Parks has indicated, respiratory injury accounts for the largest proportion of deaths in burn patients who are being managed in sophisticated burn centers.

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